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1 **MEANINGLESS IMITATION IN NEURODEGENERATIVE DISEASES:**
2 **EFFECTS OF BODY PART, BIMANUAL IMITATION, ASYMMETRY, AND**
3 **BODY MIDLINE CROSSING**

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25 **ABSTRACT**

26 Visuo-imitative apraxia has been consistently reported in individuals with dementia.
27 However, there have been substantial methodological differences across studies, and multiple,
28 sometimes competing, hypotheses have been advanced to explain action imitation deficits. Our
29 goals were to study specific imitation deficits in groups of patient participants who have been
30 selected and assigned to a group solely based on clinical criteria. We tested the effects of body
31 part, bimanual imitation, asymmetry of the model, and body midline crossing, in individuals
32 with cortical atrophy of the temporal lobes (semantic dementia, SD), frontal-parietal networks
33 (FPN, i.e., posterior cortical atrophy and corticobasal syndrome) or both (Alzheimer's disease,
34 AD). Sixty-three patient participants and 32 healthy controls were asked to imitate 45
35 meaningless finger/hand, uni-/bimanual, asymmetrical/symmetrical, and crossed/uncrossed
36 postures. While some individuals with SD were impaired for imitation of meaningless gestures,
37 individuals with FPN frequently had marked deficits across most imitation conditions.
38 Individuals with FPN tended to exhibit better performance with hand than finger postures,
39 which we argue is due to underlying visuo-constructive deficits that differentially affect finger
40 imitation. Individuals with FPN also tended to exhibit better performance with uncrossed than
41 crossed configurations, an effect which we argue is due to body schema disorganization. Some
42 individuals with AD exhibited difficulty with bimanual gesture imitation, which we argue is not
43 due to the bimanual activity itself, but rather to the complexity of the model. Overall, there were
44 significant dissociations in 34/63 patient participants (54%) across imitation sub-tests
45 (compared to controls), representing a complex pattern of dissociations according to the
46 parameters of the actions to be imitated. These findings underwrite the importance of assessing
47 meaningless imitation using stimuli that represent the full space of possible parameters of
48 imitated actions. This provides a new basis for future research to unpack which neurocognitive
49 mechanisms are disrupted to cause specific patterns of impaired imitation.

50 **KEYWORDS**

51 **Keywords:** *Alzheimer's disease, dementia, imitation, apraxia, laterality.*

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1. GOALS OF THE PRESENT STUDY

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Limb apraxia is the inability to perform intentional motor actions following brain lesions, in the absence of elementary motor disorders, sensory loss, lack of coordination, incomprehension of or inattention to commands (Geschwind, 1975; Rothi et al., 1991; Rothi et al., 1997; Signoret & North, 1979). Limb apraxia covers a wide range of impairments (Butler, 2002; Buxbaum, 1998; Buxbaum et al., 1997; Negri et al., 2007; Schwartz et al., 1991, 1995; Wheaton & Hallett, 2007) including visuo-imitative apraxia (Mehler, 1987), characterized by defective imitation of meaningless gestures (De Renzi, 1980). A diversity of error types has been documented to characterize visuo-imitative apraxia (e.g., wrong distance to the body; body side errors; finger selection errors; Bekkering et al., 2005; Goldenberg & Hagmann, 1997; Goldenberg & Karnath, 2006; Goldenberg & Strauss, 2002; Goldenberg, 1999; Goldenberg et al., 2009). Rothi et al's influential model of apraxia, and its further development, has proposed two routes for imitation: An indirect, semantic route to imitate meaningful gestures (e.g., military salute), and a direct, non-semantic route to imitate meaningful but also meaningless gestures (e.g., thumb on the ear; Achilles et al., 2016; Rothi et al., 1991; Rumiati et al., 2005, 2009, 2009; Tessari et al., 2006; Tessari & Rumiati, 2004; see also Bernardis et al., 2008; Ishibashi, 2016). Clinical double dissociations have confirmed that meaningful and meaningless imitation tap different processes (Bartolo et al., 2001; Tessari et al., 2006; see also Mengotti et al., 2013). They also depend on different brain regions. Disrupted imitation of intransitive (not tool-related) meaningful gestures has been associated with lesions of the anterior temporal and left inferior frontal lobes; Dressing et al., 2018; Weiss et al., 2016; but see also Buxbaum et al., 2014; Lesourd et al., 2018), while defective imitation of meaningless gestures has been associated with lesions of the temporo-parietal junction, posterior intraparietal sulcus, superior parietal lobule, and premotor areas; Achilles et al., 2019; Binkofski & Buxbaum, 2013; Goldenberg & Spatt, 2009; Goldenberg, 2009, 2017; Hoeren et al., 2014;

79 see also Rumiati et al., 2005; Bekkering et al., 2005). The present study focused on the imitation
80 of meaningless gestures only.

81 Test of the ability of patients to imitate meaningless actions have recently proven sensitive
82 to dementia (Dobigny-Roman et al., 1998; Lesourd et al., 2013; Li et al., 2016; Nagahama et
83 al., 2015; Ozkan et al., 2013; Sanin & Benke, 2017; Souza et al., 2016; Yamaguchi et al., 2010).
84 Previous studies have stressed that dementia may be associated with marked difficulties in the
85 imitation of bimanual gestures, as compared to unimanual gestures (e.g., Lesourd et al., 2013;
86 Sanin & Benke, 2017; Yamaguchi et al., 2010). Nevertheless, there have been substantial
87 methodological discrepancies among studies. On the one hand, little is known about how
88 different neurodegenerative diseases, associated with different patterns of cortical atrophy, may
89 affect imitation. On the other hand, there have been slight yet potentially critical differences
90 between the gestures used until now (i.e., hand Vs. finger postures; Uni- Vs. bimanual postures;
91 Asymmetrical Vs. symmetrical postures; Crossed Vs. uncrossed postures). The influence of
92 these dimensions on imitation has not been systematically addressed, which may have led to
93 misleading conclusions on the imitation skills of patients with dementia. As a result, when a
94 patient fails the meaningless imitation test, it is usually not possible to infer which dimension
95 of the test was at the source of the failure. In addition, there is still debate as to the nature of the
96 direct route for imitation. Multiple, sometimes competing hypotheses have been put forward to
97 explain visuo-imitative apraxia (see discussion below).

98 Therefore, our goal was to study specific imitation deficits in groups of patients who have
99 been selected and assigned to a group solely based on clinical criteria. This study aimed at
100 determining which dimensions of meaningless imitation are at the origin of visuo-imitative
101 apraxia (i.e., finger/hand, uni-/bimanual, asymmetrical/symmetrical, crossed/uncrossed
102 postures), in patients with cortical atrophy of temporal lobes (i.e., semantic dementia), frontal-
103 parietal lobes (i.e., corticobasal syndrome, posterior cortical atrophy), or both (i.e., Alzheimer's

104 disease). Our secondary goal was to infer the cognitive correlates of meaningless imitation
105 impairments in this population, in the light of the hypotheses available in the apraxia literature.

106 **2. THEORETICAL BACKGROUND**

107 *2.1. LIMB APRAXIA IN NEURODEGENERATIVE DISEASES*

108 Limb apraxia is a frequent condition in neurodegenerative diseases (e.g., Buchmann et
109 al., 2020), like posterior cortical atrophy (PCA; Benson et al., 1988; Crutch et al., 2017) and
110 corticobasal syndrome (CBS; Armstrong et al., 2013; Litvan et al., 1997). The emergence of
111 non-amnesic cognitive phenotypes of Alzheimer’s disease (AD) in recent criteria (McKhann
112 et al., 2011) has raised the importance of a thorough examination of higher-order motor
113 symptoms in neurodegenerative diseases. Literature on this topic is, however, sparse in
114 comparison with other cognitive domains (Lesourd et al., 2013). Posterior cortical atrophy is
115 clinically characterized by a progressive decline of visual-spatial skills, sometimes associated
116 with limb apraxia but contrasting with the relative preservation of memory (Benson et al., 1988;
117 Crutch et al., 2012, 2013; McMonagle et al., 2006). Whereas episodic memory impairments
118 dominate the clinical picture in “typical” AD, PCA patients with AD pathology fail
119 neuropsychological tasks that test functions that depend on fronto-parietal areas. Corticobasal
120 degeneration is an atypical parkinsonian syndrome that is characterized by extrapyramidal (e.g.,
121 limb rigidity, bradykinesia, abnormal gait, postural instability, falls, tremor) as well as higher
122 cortical signs typically associated with lesions of frontal and parietal networks, like limb apraxia
123 (Armstrong et al., 2013;) executive dysfunction (Pillon et al., 1995), or visual-spatial
124 impairments (Possin, 2010). Limb-kinetic apraxia is a form of apraxia affecting finger dexterity,
125 which may differentially hamper finger imitation; Denes et al., 1998; Gross & Grossman, 2008;
126 Leiguarda et al., 2002; Luria, 1978). So, PCA and CBS have been associated with both lesions
127 of parietal lobes and limb apraxia. In contrast, visuo-imitative apraxia is generally not observed

128 in patients with semantic dementia (SD; Neary et al., 1998; see also “semantic variant of
129 primary progressive aphasia”, Gorno-Tempini et al., 2011). The latter typically have bilateral
130 anterior temporal lobe lesions resulting in marked impairments of semantic memory (e.g., loss
131 of word or object meaning and comprehension, impaired naming, surface dyslexia or
132 dysgraphia, semantic paraphasias, associative agnosia, prosopagnosia), contrasting with
133 relative sparing of linguistic abilities and episodic memory; Gorno-Tempini et al., 2011; Neary
134 et al., 1998; Snowden et al., 2018; see also Blundo et al., 2006).

135 Given the clinical characteristics of the abovementioned groupings of patients (Felician
136 et al., 2003; Seeley et al., 2009), we combined the patients with either posterior cortical atrophy,
137 or corticobasal syndrome, into a Frontal-Parietal Network (FPN) group. Those individuals were
138 expected to demonstrate imitation deficits. In contrast, patients with semantic dementia were
139 expected to perform in normal range assuming that parietal and frontal brain regions and
140 functions are spared in most patients (Hodges et al., 1992; Kramer et al., 2003). The
141 performance of patients with Alzheimer’s disease was expected to be in an intermediate position
142 between the two abovementioned groups, for these patients generally have temporal lobe
143 lesions with or without marked parietal lobe lesions depending on cases (Braak & Braak, 1991,
144 1997; Seeley et al., 2009). So, as a group, they were expected to show less severe imitation
145 impairments than patients with CBS or PCA.

146 2.2. *THE MOTOR AND COGNITIVE BASES OF MEANINGLESS IMITATION*

147 Previous studies have documented at least four dissociable aspects of meaningless
148 imitation that might reveal different underlying motor-cognitive impairments. Table 1
149 summarizes the core hypotheses that may account for these effects, as well as the related
150 predictions.

151 2.2.1 The body part effect

152 The *body part effect* corresponds to behavioral dissociations between finger and hand
153 postures (Goldenberg & Karnath, 2006; Goldenberg & Strauss, 2002; Goldenberg, 1995, 1999;
154 Goldenberg et al., 2009; see also Rumiati et al., 2005; Tessari et al., 2006). Fingers and hands
155 are part of body representations, and are represented by both differences between body
156 structures (e.g., the nose differs from the chin, the index finger differs from the little finger),
157 and differences in their relative localization (e.g., nose over the chin, index finger of the right
158 hand to the left of the little finger). Yet, clinical studies have suggested that finger and hand
159 imitation may put different loads on cognitive functions. Goldenberg and colleagues have
160 provided three empirical arguments for this dissociation. First, there are clinical dissociations
161 between hand and finger postures in patients with stroke (Goldenberg, 1999; Goldenberg &
162 Strauss, 2002). Second, patients with imitation impairments may also fail to reproduce
163 configurations on a manikin, suggesting an involvement of body representations in the coding
164 of body parts in both the first and third person perspective (Goldenberg, 1995). Third, there is
165 a positive association between impaired imitation of finger configurations, and impaired visual-
166 spatial skills (Goldenberg et al., 2009). So, even though both fingers and hands are represented
167 in the body map, imitating finger or hand configurations may put different loads on different
168 cognitive processes, as explained by Goldenberg (2001): “*Hand postures are determined by*
169 *relationships between a considerable number of different body parts, like lips, nose, ear, chin,*
170 *cheek, back of hand, or palm of hand. Knowledge about the classification and boundaries of*
171 *all of these parts is needed for coding of hand postures. In contrast, finger postures concern*
172 *only a uniform set of five body parts, and the conceptual distinction between them is largely*
173 *exhausted by an appreciation of their serial position. Body-part coding of hand postures may*
174 *therefore [...] demand more knowledge about the structure of the human body than coding of*
175 *finger postures. [...] Fingers are perceptually similar, and their identity is mainly determined*
176 *by their spatial position with respect to other fingers. A distinction between, for example, the*

177 *middle and the ring finger is likely to put higher demands on visuospatial exploration than a*
178 *distinction between the edge and the palm of the hand or between the cheek and the forehead.*
179 *Thus, perceptual analysis is probably more difficult for finger than for hand postures”*
180 (Goldenberg, 2001, p. S134).

181 In this framework, imitation of hand postures directed toward the body, performed
182 without visual control, relies on the perceptual discrimination of body parts (e.g., chin, lips,
183 nose, ears) and hence may call for body representations (i.e., the *body representation*
184 *hypothesis*; Goldenberg, 1995). Body representations encompass both body image (i.e., general
185 knowledge about the structure of the body; Goldenberg, 1995) and body schema (i.e., the
186 tridimensional representation of the body personal and peripersonal space during action;
187 (Buxbaum, 2001; Schwoebel & Coslett, 2005; Sirigu et al., 1991). According to the body
188 representation hypothesis (Goldenberg, 1995; Schwoebel et al., 2004; Schwoebel & Coslett,
189 2005; Sirigu et al., 1991), marked difficulties to imitate hand, but not finger postures, may
190 reveal disorganization of body representations.

191 In contrast, imitation of finger configurations may put higher demands on visual-spatial
192 and executive control due to the visual similarity of fingers, suggesting that finger imitation is
193 similar to classical visuoconstructive tests (i.e., *the visuo-constructive hypothesis*; Goldenberg,
194 2001; Lesourd et al., 2013). In line with this proposal, finger imitation is particularly impaired
195 in patients with right parietal lobe lesions causing visual-spatial impairments and neglect
196 (Goldenberg & Strauss, 2002; Goldenberg, 1999). According to the visuo-constructive
197 hypothesis, marked difficulties to imitate finger but not hand postures should be observed and
198 may reveal either visual-spatial or executive dysfunction (or both). If the visuo-constructive
199 hypothesis is correct, the imitation performance should also be a function of the number and
200 spatial complexity of visual elements. It is expected that bimanual and asymmetrical postures
201 are more difficult to imitate than unimanual and symmetrical postures. We further assume that

202 visual-spatial processing contributes to imitation, especially of bimanual and asymmetrical
203 postures. Subsequently, bimanual and asymmetrical postures were expected to be more difficult
204 to imitate than unimanual and symmetrical postures, respectively. Finally, positive correlations
205 were expected between classical visual-spatial and executive tests on the one hand, and
206 imitation of finger, bimanual and/or asymmetrical postures on the other hand.

207 2.2.2 The bimanual effect

208 In dementia studies, emphasis has been placed on bimanual imitation. The *bimanual effect*
209 manifests itself in the higher difficulty of bimanual postures as compared with unimanual
210 postures, documented in Alzheimer's disease, mild cognitive impairments, or dementia with
211 Lewy bodies (Lesourd et al., 2013; Li et al., 2016; Nagahama et al., 2015; Sanin & Benke,
212 2017; Yamaguchi et al., 2010). This pattern has resulted in the creation of a new "bimanual
213 apraxia" category (Sanin & Benke, 2017) characterized by combined motor, spatial, and
214 executive impairments of bimanual gesture imitation in patients with Alzheimer's disease.
215 Whether bimanual apraxia is specific to Alzheimer's disease, or can also be observed in focal
216 cortical atrophy syndromes, remains an open question. More importantly, it is not known
217 whether bimanual apraxia arises from bimanual activity per se, because there has not been
218 systematic comparison of uni- and bimanual imitation skills, controlling for other dimensions
219 of the test (i.e., body part, asymmetry, or midline-crossing). For example, most studies on
220 bimanual imitation have used either finger configurations only, or mixed lists of hand and finger
221 items. The heterogeneity of evaluation methods may explain why there is still controversy as
222 to whether patients with dementia do have early bimanual imitation impairments (Li et al.,
223 2016; Sanin & Benke, 2017; Yamaguchi et al., 2010). Thus, one of the goals of the study was
224 to verify the prevalence of bimanual apraxia (i.e., "unimanual > bimanual" dissociations) while
225 controlling for other dimensions of the test.

226 2.2.3 The asymmetry effect

227 The *asymmetry effect* corresponds to the increased difficulty of asymmetrical gestures, in
228 comparison with symmetrical ones (Ajuriaguerra et al., 1966; Huhn et al., 2014; Kelso et al.,
229 1979). Some early studies have reported it in patients with Alzheimer’s disease (Ajuriaguerra,
230 1960; Ajuriaguerra et al., 1966), but there are no data on other syndromes. Asymmetrical
231 gestures can be defined as gestures for which motor patterns (e.g., the selected fingers) or goals
232 (e.g., the hand-body contact point) are not the same for each hand. Humans have a natural
233 preference for functional (goal) and timing symmetry during bimanual tasks (Huhn et al., 2014;
234 Kelso et al., 1979) while the spatial parameters and motor innervation patterns of each upper
235 limb may vary. This has suggested that large groups of muscles act as functional units (thus
236 simplifying movement control), reflecting the existence of a bilateral motor control system
237 (Brinkman & Kuypers, 1973; Kinsbourne, 2003). Healthy adults, however, are able to break
238 bimanual synergies – an ability needed to play a musical instrument or drive a car – through
239 initiation strategies (i.e., delaying limb activations) or inhibitory neural networks (Baumard et
240 al., 2020; Swinnen et al., 1991; see also Wallon & Lurçat, 1962). This in turn suggests that
241 difficulties in desynchronizing hand movements to imitate asymmetrical configurations may
242 reveal abnormal motor cognition.

243 Performing asymmetrical gestures requires selection and organization of the parameters
244 governing the action across both effectors. According to the *goal-directed theory of imitation*
245 (Bekkering et al., 2000, 2005; Gleissner et al., 2000), imitation results from the combination of
246 elementary action patterns (subgoals) into more general action patterns (goals), with
247 hierarchically organized goal structure dissociating means and ends (e.g., one must identify the
248 end goal “reaching the ear” before identifying the subgoal “reaching the left ear”, and the mean
249 “with the left hand”). Left hemisphere lesions have been proposed to affect the ability to bind
250 and maintain goals into such a hierarchical structure, resulting in simplifications of the model

251 (Bekkering et al., 2005). This theory suggests that the activity of each hand is bound into one
252 and the same general goal during bimanual imitation of symmetrical gestures (e.g., both hands
253 target the ears), while it cannot be the case regarding asymmetrical gestures (e.g., one hand
254 targets the ear while the other one targets the neck). In this framework, impaired imitation of
255 asymmetrical gestures (i.e., different goals for each hand) but not of symmetrical gestures (i.e.,
256 similar goals) may be interpreted as a disruption of the hierarchical goal structure. In this view,
257 one should also observe simplifications, i.e. a tendency to make symmetrical postures when
258 presented with an asymmetrical model. To our knowledge, the available literature does not
259 allow one to predict the reverse “asymmetrical > symmetrical” pattern.

260 2.2.4 The midline-crossing inhibition effect

261 Healthy individuals have a natural tendency to use the hand of the same body side as the
262 target (Furlanetto et al., 2014; Yamamoto & Kitazawa, 2001), yet they still have the ability to
263 override this left-right organization of the reaching space. The *midline crossing inhibition effect*
264 manifests itself in difficulties to reach, step, or look across the body midline, thus resulting in
265 difficulties to imitate contralateral gestures (e.g., touching the left ear with the right index
266 finger). It has been documented in stroke patients (Head, 1920, 1926, cited by Bekkering et al.,
267 2000), in typically developing children (“crosslateral” or “contralateral” inhibition effect;
268 Bekkering et al., 2000; Gleissner et al., 2000; Gordon, 1923; Lombardi et al., 2000; Schofield,
269 1976; Wagner & Cirillo, 1968; Wallon & Lurçat, 1962), as well as in healthy adults (Baumard
270 et al., 2020; Pedersen et al., 2005), but barely described in patients with dementia, to our
271 knowledge. Previous reports have suggested (but not demonstrated) that crossed hand
272 configurations (i.e., one or both hands cross the body-midline axis and encroach(s) on the
273 contralateral space) may be more difficult to imitate than uncrossed configurations (i.e., each
274 hand remaining in the ipsilateral side; Ajuriaguerra, 1960; Yamaguchi et al., 2010) for patients
275 with dementia. In the framework of the body representation hypothesis, midline-crossing

276 inhibition has been proposed to reflect insufficient perceptual-motor conversion mechanisms
277 underlying body representation (Ajuriaguerra, 1960; Ajuriaguerra et al., 1966; Buxbaum, 2001;
278 Gooddy & Reinhold, 1952; Kephart, 1971). In the framework of the goal-directed theory of
279 imitation, Gleissner et al. (2000) have rather assumed that midline crossing is coded at a low
280 level of movement goal hierarchy. In this view, the end-point of the action (e.g., hand on the
281 ear) is more important than other parameters like the hand used (i.e., left/right) or the need to
282 cross the body midline. This may explain simplification errors (e.g., reaching the correct target
283 “ear” but without crossing the midline). Both hypotheses predicted that performance with
284 imitation of crossed gestures would be more impaired than imitation of uncrossed gestures,
285 while the available literature does not allow the reverse prediction to our knowledge.

286 **3. MATERIALS AND METHODS**

287 *3.1. PARTICIPANTS*

288 Thirty-two healthy controls and 63 patients diagnosed with Alzheimer’s disease (AD),
289 semantic dementia (SD), posterior cortical atrophy (PCA), or corticobasal syndrome (CBS)
290 took part in the study (Table 2). All patients were recruited in four French outpatient
291 neurological units (i.e., Angers, Rennes, Lyon, Grenoble). Patients were diagnosed by
292 experienced neurologists with neurological examination, neuropsychological assessment,
293 imaging data and biomarker analysis. They met criteria for probable Alzheimer’s disease of the
294 amnesic form (McKhann et al., 2011), semantic dementia (Gorno-Tempini et al., 2011; Neary
295 et al., 1998), posterior cortical atrophy (Benson et al., 1988; Crutch et al., 2017; Renner et al.,
296 2004; Tang-Wai et al., 2004), and corticobasal syndrome (Armstrong et al., 2013; Litvan et al.,
297 1997). Exclusion criteria included having a history of either neurological or major psychiatric
298 illness; having a rheumatologic condition, mood disorder, medical treatment or cognitive state
299 incompatible with the protocol; or a MMSE score below 10. Two PCA patients and one CBS

300 patient were finally excluded from the sample because of depressive symptoms ($n = 2$) or
301 diagnostic uncertainty ($n = 1$). In order to avoid exaggerating deficits by comparing older
302 patients to young adults, healthy controls were matched on age and sex only to the oldest
303 patients that is, patients with Alzheimer's disease. The study followed the guidelines of the
304 Declaration of Helsinki and was approved by local ethics committee (Western Protection to
305 Persons Committee II, n° 2012/32). There was substantial sample overlap with previous tool
306 use studies (Baumard et al., 2016, 2018). The healthy control group was part of a larger sample
307 ($n = 103$) from a previous imitation study (Baumard et al., 2020) while the clinical data on
308 imitation are original.

309 3.2. *NEUROPSYCHOLOGICAL ASSESSMENT*

310 In addition to routine cognitive tests, all patients underwent neuropsychological
311 assessment. General cognitive functioning was tested using the French Battery of Cognitive
312 Efficacy (Signoret et al., 1989). This battery consists of eight tests of working memory (i.e.,
313 listing the days of the week in reverse order), orientation (i.e., questions on age, year, month,
314 date, name of the president of the republic), verbal learning (i.e., immediate recall of eight
315 concrete words across 3 trials), visual recognition (i.e., immediate and delayed recall, then
316 recognition of six black and white pictures among three foils each), verbal reasoning (i.e.,
317 similarities, arithmetic problem, and proverb definition), verbal animal fluency, naming of 12
318 black and white pictures, and visuoconstructive skills (i.e., copying a 3-D cube, and three 2-D
319 overlapping triangles). Each subtest is scored on a 12-point scale with any score below 9 being
320 suggestive of cognitive impairment. The maximum score is 96 (i.e., 12 points x 8 subtests = 96)
321 with a cut-off score of 80 – corresponding to -1.96 standard deviations from the mean. These
322 values are based on French normative data (control sample, $n = 30$ participants 65 to 89 years
323 of age).

324 Executive functioning was measured using the fast Frontal Assessment Battery (FAB;
325 Dubois et al., 2000) that tests conceptualization (i.e., similarities), mental flexibility (i.e., letter
326 fluency), programming (i.e., motor series of Luria), sensitivity to interference (i.e., conflicting
327 instructions), inhibitory control (i.e., go-no-go), environmental autonomy (i.e., prehension
328 behavior). Each subtest is scored on a 3-point scale for a maximum score of 18 (i.e., 3 points x
329 6 subtests). Any score below or equal to 15 (corresponding to -1.96 standard deviations from
330 the mean) may indicate cognitive impairment based on French normative data (control sample,
331 n = 42 participants, mean age 58, standard deviation 14.4).

332 The results were consistent with the diagnosis as the core symptoms were orientation and
333 memory disorders in Alzheimer's disease, verbal-semantic impairments in semantic dementia,
334 and visuoperceptive deficits in posterior cortical atrophy (Table 2). Patients with corticobasal
335 syndrome had relatively spared cognitive functioning and were diagnosed on the basis of
336 extrapyramidal symptoms and upper limb apraxia (e.g., limb-kinetic apraxia). The imitation
337 tasks described below were not used to establish the diagnosis.

338 3.3. *IMITATION OF MEANINGLESS GESTURES*

339 3.3.1. Materials and procedure

340 This task was inspired from the ones used by Goldenberg (Goldenberg & Hagmann, 1997;
341 Goldenberg, 1999) and Peigneux and Van der Linden (2000). Unimanual postures were the
342 same as in Goldenberg & Hagmann (1997) with the exception of one item, because of a ceiling
343 effect found in pretests (i.e., "palm down over the head" became "hand vertical on the head,
344 thumb oriented downward"). The full list of gestures is available in Figure 1 and Supplementary
345 Figure 1. Participants sat in front of the examiner, and were asked to successively imitate 45
346 meaningless configurations in a mirror-like fashion (i.e., axial rather than central symmetry).
347 They used their dominant hand, and they could see the model as they imitated (i.e., concurrent
348 imitation). Gestures varied on the following criteria: body parts (i.e., finger/hand), number of

349 hands (i.e., unimanual/bimanual), symmetry of configurations (i.e.,
350 symmetrical/asymmetrical), and body-midline crossing (i.e., crossed/uncrossed). Importantly,
351 gestures were selected or created so that they varied only on the abovementioned independent
352 variables, when possible (i.e., when it was consistent with biomechanical constraints and when
353 it allowed creating unambiguous items). So, bimanual symmetrical configurations were derived
354 from unimanual configurations by duplicating the initial posture (e.g., thumb-index ring
355 became two thumb-index rings interlaced). Bimanual asymmetrical configurations consisted of
356 slight alterations of symmetrical ones. The same rationale was applied to crossed and uncrossed
357 configurations. As in previous studies, the participants could see their own hand during the
358 imitation of finger configurations, whereas hand configurations were directed toward the head
359 and neck so that the participant could not rely on visual control (Goldenberg & Haggmann,
360 1997). The task order was fixed and was the same as in Figure 1 (the exact sequence is available
361 in Supplementary Figure 1). The item order within each task was also the same for all
362 participants. Bimanual postures may be more difficult to imitate than unimanual postures for
363 healthy controls, based on previous studies (Baumard et al., 2020). Therefore, we set different
364 time limits for unimanual and bimanual postures to control for intrinsic task difficulty and avoid
365 floor effects. Based on our data (n = 103), healthy adults needed less than 10 seconds to imitate
366 unimanual postures in 99.9% of observations, and less than 20 seconds to imitate bimanual
367 postures in 99.5% of observations. Therefore, the time limit was set to 10 seconds for unimanual
368 postures and 20 seconds for bimanual postures, to avoid a floor effect in the latter condition.
369 For the same reason, two practice items preceded each series of bimanual postures (i.e., one
370 finger configuration and one hand configuration, both symmetrical).

371 3.3.2. Quantitative data

372 Performance was video-recorded and later coded on a 2-point scale similar to those used
373 in previous studies (e.g., Baumard et al., 2020; Goldenberg & Haggmann, 1997; Souza et al.,

2016). Perfect imitation of the model within the time limit was worth 2 points, while approximate imitation within the time limit was worth 1 point (e.g., correct hand position on the body but incorrect hand orientation). No point was awarded in the absence of performance or if the imitation obviously differed from the model. Hesitations and self-corrections were accepted because they were frequent in healthy adults. Thereby performance was scored only when participants stopped trying new configurations and held the gesture for at least two seconds. The coding system focused on the position of fingers in the “finger” items, and on the position and orientation of the hand relative to the body in the “hand” items. This coding system has been used to test dissociations between the imitation of hand and finger configurations in stroke patients (Goldenberg & Hagmann, 1997). As in previous studies, finger postures were expected to put higher loads on visual-spatial skills whereas hand postures were expected to put higher loads on body representations (see the rationale behind this in section 2.2.1). We used a similar coding system because our goal was to test how the body part effect documented in stroke patients would apply to patients with neurodegenerative diseases. The maximum total score was 90 (i.e., 45 items x 2 points).

Two coders tested the scoring system on 4 videos (1 participant from each group). Then, they coded separately 30% of the videos (i.e., 10 HC, 10 AD, 5 SD, and 5 FPN participants). One of the coders was unaware of the hypotheses and of the clinical status of the participants. The two raters agreed on 1109 out of 1350 observations (i.e., 45 observations x 30 participants) so the mean percentage of agreement at the item level was 82.2%. The correlation based on the total imitation score was high ($r = 0.96$, $p < .001$) and similar to the ones found in previous studies (e.g., Li et al., 2016; Sanin & Benke, 2017). Cohen’s Kappa was calculated (with scores of 2, 1, or 0 considered as categories) since it adjusts for the level of agreement that would occur by chance. With this method, inter-rater reliability was moderate ($K = 0.59$).

3.3.3. Error analysis

399 In an attempt to better describe the performance (see also Bekkering et al., 2005), we
400 defined two possible errors in bimanual subtests: Symmetry errors and body-midline crossing
401 errors.

402 Symmetry errors were counted only in the 10 asymmetrical items and were defined as
403 follows: At any moment of the performance, the participant generates a symmetrical
404 configuration, whether it concerns either the selection of fingers (in the “finger” items) or the
405 position of the hands on the body (in the “hand” items). This type of error was thought to reflect
406 difficulties in inhibiting bimanual synchronization. There were 10 items (5 “finger” and 5
407 “hand” items). One point was given for each error (1 max. per item). Therefore, the maximum
408 number of errors was 10 (5 for “finger” items and 5 for “hand” items).

409 Body-midline crossing errors were taken into account only in the 10 bimanual “crossed”
410 items. They were defined as follows: At any moment of the performance, the participant
411 generates a bimanual configuration directed toward the body, that does not cross the body
412 midline. This type of error was thought to reflect difficulties in overriding the left-right
413 organization of the peripersonal space. There were 10 items (all of which were “hand” items).
414 One point was given for each error (1 max. per item). Therefore, the maximum number of errors
415 was 10.

416 3.4. STATISTICS

417 Raw imitation scores (but not error counts) were converted into percentages of the
418 maximum score for the sake of clarity (e.g., a score of 7/10 was converted into 70%). Since the
419 different imitation conditions varied in difficulty (Baumard et al., 2020), raw scores were also
420 converted into t-scores. Individual deficits and dissociations were tested using modified t-tests
421 (with a two-tailed probability) controlling for the mean, standard deviation and between-task
422 correlations observed in healthy controls (Crawford & Garthwaite, 2002, 2005;
423 Singlims_ES.exe and Dissocs_ES.exe softwares). This method allows the detection of

424 dissociations between tasks that are rare in the control population. As a result, the dissociations
425 observed in individual patients can be interpreted as an effect of the patient's clinical/cognitive
426 status. The frequency of these dissociations was compared between groups with Chi-square
427 tests, or Fisher tests in case of low values. Unless otherwise specified, p -values are corrected p -
428 values using Holm's method for multiple tests. "Tendencies toward significance" were reported
429 if p -value was no longer significant after p -value adjustment. Finally, we performed a multiple
430 regression analysis of the cognitive scores contributing to the variance of imitation scores.

431 **4. RESULTS**

432 *4.1. FREQUENCY AND SEVERITY OF IMITATION IMPAIRMENTS*

433 Individual raw scores and t-scores are available in Supplementary tables 1 and 2. We
434 found 19 different patterns of imitation performance (Supplementary Table 3). Given this large
435 number of profiles, we first studied how the presence/absence of imitation deficits mapped the
436 clinical groups. As shown in Table 3, 31% of SD cases (5/16), 47% of AD cases (15/32), and
437 87% of FPN cases (13/15) failed at least one of the imitation tests. The difference of distribution
438 was significant between the SD and FPN groups ($\chi^2 = 7.62$, $df = 1$, $p = .017$), between the AD
439 and FPN groups ($\chi^2 = 5.16$, $df = 1$, $p = .046$), but not between the SD and AD groups ($\chi^2 = 0.52$,
440 $df = 1$, $p = .469$).

441 We also studied the severity of the imitation deficit, by studying the distribution of cases
442 as a function of the number of imitation conditions impaired (i.e., 0, 1-2, 3-4, 5-6, 7). The dis-
443 tribution, shown in Table 3, varied between the SD and FPN groups (Fisher test, $p < .001$), as
444 well as between the AD and FPN groups ($p < .001$), but not between the SD and AD groups
445 (Fisher test, $p = .080$). Failure to imitate any of the seven conditions was observed only in the
446 FPN group. Failure to imitate at least one of the two unimanual conditions (i.e., the easiest

447 conditions for healthy controls, see Table 4) was observed in 6% of SD cases (1/16), 28% of
448 AD cases (9/32), and 87% of FPN cases (13/15). Again, the distribution was different between
449 the SD and FPN groups ($\chi^2 = 17.10$, $df = 1$, $p = <.001$), between the AD and FPN groups ($\chi^2 =$
450 11.80 , $df = 1$, $p = .001$), but not between the SD and AD groups (Fisher test, $p = .132$).

451 4.2. *IMITATION DEFICITS AS A FUNCTION OF GROUP AND CONDITION*

452 Table 4 emphasizes significant control/patient differences as a function of imitation
453 conditions (see also Supplementary Figure 2). To sum up, patients from the SD group had
454 normal to subnormal imitation performance, whereas patients from the FPN group had frequent
455 and important imitation impairments. Difficulties to imitate unimanual postures were
456 particularly frequent and pronounced in the FPN group. Patients from the AD group either
457 resembled SD patients, or were in an intermediate position between the SD and FPN groups
458 (Supplementary Table 3). They showed imitation impairments mainly with bimanual finger
459 asymmetrical postures as well as with bimanual hand configurations that were asymmetrical
460 and required midline crossing.

461 4.3. *DISSOCIATIONS BETWEEN CONDITIONS*

462 Given the presence of both group effects and within-group heterogeneity, condition ef-
463 fects should be studied group by group, but also in individual cases, because opposite dissoci-
464 ations may exist in different patients from the same clinical group (e.g., case X may perform
465 better with finger than hand postures while case Y may show the reverse pattern). Therefore,
466 we studied dissociations in individual cases independently from the clinical group of origin,
467 and then we tested whether the expected dissociations were more frequent in some clinical
468 groups than in others (Table 5; Supplementary Table 4). Of note, we tested dissociations based
469 on raw scores, but we concluded on the direction of the dissociation (i.e., score $X < Y$ or score
470 $X > Y$) based on t-scores, in order to control for intrinsic task difficulty. As a result, a patient
471 may have a higher raw score in condition X than in condition Y, and yet show an “ $X < Y$ ”

472 dissociation based on the variance observed in the healthy control group. This method allows
473 controlling for variations in difficulty across imitation conditions.

474 Overall, 54% of the patient sample (34/63) showed at least one of the expected dissocia-
475 tions: 5 SD cases (31%), 18 AD cases (56%), and 11 FPN cases (73%). Each of the four effects
476 of interest are reported below.

477 4.3.1. The body part effect

478 We found 18 dissociations (observed in 13 cases) between finger and hand postures (ei-
479 ther uni- or bimanual): 17 “Finger < Hand” dissociations (among which 8 were strong dissoci-
480 ations), and 1 “Finger > Hand” dissociation. There were more cases showing a “Finger < Hand”
481 dissociation in the FPN group ($n = 7/15$) than in the SD group ($n = 1/16$; Fisher test, $p = .046$).

482 4.3.2. The bimanual effect

483 We found 28 dissociations (observed in 21 cases) between unimanual and bimanual pos-
484 tures (either finger or hand): 21 “Unimanual < Bimanual” dissociations (among which 12 were
485 strong dissociations), and 7 “Unimanual > Bimanual” dissociations (among which one was a
486 strong dissociation). The “Unimanual > Bimanual” dissociation was observed in 5/32 AD cases
487 (i.e., 16%) and 2/16 SD cases (13%), and there was no difference between the SD and FPN
488 group ($p = 0.484$). The unexpected “Unimanual < Bimanual” dissociation was more frequent
489 in the FPN group ($n = 10/15$) than in the SD group ($n = 1/16$, Fisher test, $p = .008$).

490 Since only one hand was tested in the unimanual condition, one could argue that right-
491 left asymmetry of motor functions, which is frequent in patients with corticobasal syndrome,
492 may have exaggerated this dissociation between unimanual and bimanual imitation in the FPN
493 group. Nevertheless, this dissociation was found in 6 cases with posterior cortical atrophy, who
494 do not have motor symptoms. In the 4 remaining CBS cases, imitation scores were available
495 for both the right and left hand. We calculated the raw difference between the imitation scores

496 obtained with the right and left hands (e.g., if a patient has a score of 80/100 with the right hand,
497 and 70/100 with the left hand, the difference score is +10). In the control sample, the raw dif-
498 ference between the score obtained with the right and left hands ranged from -10 to +5 for finger
499 configurations, and from -5 to +5 for hand configurations (on a 0-100 scale). Two cases showed
500 an abnormal right-left difference, with hand configurations only (CBS.02: -10; CBS.08: -10).
501 Yet, neither of these two cases showed the abovementioned dissociation between unimanual
502 and bimanual imitation. In contrast, 2 CBS cases showed dissociations between unimanual and
503 bimanual imitation, but no dissociation between the right and left hand (CBS.01 and 06). This
504 suggests that differences between imitation scores of the right and left hands did not exaggerate
505 the prevalence of the dissociation between unimanual and bimanual dissociation.

506 4.3.3. The asymmetry effect

507 We found 14 dissociations (observed in 14 cases) between asymmetrical and symmetrical
508 postures (either finger or hand): 11 “Asymmetrical < Symmetrical” dissociations (classical dis-
509 sociations only), and 3 “Asymmetrical > Symmetrical” dissociations (among which 2 were
510 strong dissociations; Table 5, Supplementary Table 4). The distribution of dissociations did not
511 vary across the SD and FPN groups ($p = .452$).

512 4.3.4. The midline crossing inhibition effect

513 We found 11 dissociations (observed in 11 cases) between crossed and uncrossed pos-
514 tures. There were only “Crossed < Uncrossed” dissociations (among which 2 were strong dis-
515 sociations). There were more cases showing the “Crossed < Uncrossed” dissociation in the FPN
516 group ($n = 7/15$) than in the SD group ($n = 0/16$; Fisher test, $p = .009$).

517 *4.4. ERROR ANALYSIS*

518 The number of symmetry errors (finger configurations) was virtually the same in the HC
519 (mean = 1.6, sd = 0.8), SD (mean = 1.9, sd = 1.2), AD (mean = 2.1, sd = 1.2), or FPN groups

520 (mean = 1.9, sd = 1.3; $F(3, 90) = 1.01, p = .39$). The same was found with hand configurations
521 (HC mean = 0.9, sd = 0.8; SD mean = 0.9, sd = 0.6; AD mean = 1.3, sd = 1.0; FPN mean = 1.0,
522 sd = 1.2; $F(3, 91) = 1.03, p = .38$).

523 HC participants committed fewer midline crossing errors (mean = 0.2, sd = 0.5) than SD
524 (mean = 0.9, sd = 1.1), AD (mean = 0.8, sd = 1.0), or FPN patients (mean = 1.7, sd = 1.6). There
525 was a main effect of GROUP ($F(3, 91) = 6.99, p < .001$). Post-hoc analyses confirmed the
526 HC/FPN difference ($t(15.1) = -3.36, p < .001$) only. One HC participant made 2 errors, 5 par-
527 ticipants made 1 error, and 26 made no error. In contrast, 8 FPN participants made 2 or more
528 errors, 2 participants made 1 error, and 5 participants made no error.

529 4.5. RELATIONS OF IMITATION TO NEUROPSYCHOLOGICAL SCORES

530 In order to infer predictors of imitation, we compared subgroups of patients as a function
531 of the number of imitation conditions impaired (Table 3). We used the following dependent
532 variables: the MMSE score as a measure of global cognitive decline; the FAB score as a meas-
533 ure of executive functioning; and the visuoconstructive test of the BEC96 battery as a measure
534 of visuoconstructive skills. We selected these variables with regard to the visuoconstructive
535 hypothesis, and to control for global cognitive decline. We performed these analyses regardless
536 of the clinical group. As displayed in Table 3, the higher the number of imitation conditions
537 impaired, the lower the cognitive scores – especially the visuoconstructive score. We used the
538 same method to study the cognitive scores of cases showing the dissociations of interest (Table
539 5). Based on data observation, cases with a “Finger < Hand” dissociation had lower visuocon-
540 structive scores than other cases (a Mann-Whitney U test was preferred given the low number
541 of cases; $U = 85.0, p < .001$).

542 The results of the multiple regression analysis (Table 5) also confirm that the visuocon-
543 struction score is overall a better predictor of imitation scores than the MMSE and executive

544 (FAB) score. Plots are available in Supplementary Figure 3. We could not confirm that this
545 relation was due to the performance of the FPN group, given the low sample sizes. Future stud-
546 ies with larger samples and more statistical power should test the covariance relations within
547 each clinical group.

548 **5. DISCUSSION**

549 The goals of this study were to describe imitation deficits in independently defined
550 clinical groups of patients with neurodegenerative diseases. A secondary goal was to study the
551 nature of cognitive disruption that can give rise to visuo-imitative apraxia in patients with
552 neurodegenerative diseases. In our findings, visuo-imitative apraxia was rare in patients with
553 semantic dementia (SD), whereas it was both frequent and pronounced in patients from the
554 “Frontal-Parietal Network” (FPN) group. These findings are in line with previous lesion studies
555 on stroke patients that have demonstrated that fronto-parietal lesions are associated with deficits
556 in imitating meaningless actions (Binkofski & Buxbaum, 2013; Buchmann & Randerath, 2017;
557 Buxbaum, 2017; Dressing et al., 2018; Goldenberg & Spatt, 2009; Goldenberg, 2017; Mengotti
558 et al., 2013). Patients with Alzheimer’s disease (AD) showed a more nuanced picture, probably
559 reflecting clinical heterogeneity but also patient selection: Contrary to previous studies, we have
560 included “typical” amnesic AD and patients with posterior cortical atrophy (PCA) in separate
561 groups based on the ventral-dorsal model of imitation. That said, the performance varied as a
562 function of conditions. This confirms that meaningless gestures are not monolithic: Slight
563 changes in the model performing the action may lead to substantial differences in performance
564 across patients, both within and across clinical groups. This may explain why the meaningless
565 imitation skills of AD patients have been subject to large variations in previous studies (from
566 10% to 100% of the maximum score; see Lesourd et al., 2013). The next sections will, therefore,
567 focus on the effects of imitation conditions.

568 *5.1. THE BODY PART EFFECT*

569 While individual “Finger > Hand” dissociations were rare and inconsistent across
570 conditions, “Finger < Hand” dissociations were observed in 13 cases (i.e., 21% of the clinical
571 sample). They were more frequent in the FPN group than in the SD group, suggesting that this
572 dissociation was relatively specific to patients with lesions of fronto-parietal areas. These
573 dissociations were also observed in the AD group, mainly with bimanual postures, which is
574 consistent with previous studies (Johnen et al., 2015). Of note, Sanin and Benke (2017) found
575 similar levels of finger and hand impairments in Alzheimer’s disease, but they compared static
576 finger postures and alternate hand movements; by using static postures only, and by controlling
577 the asymmetry of the model, we found that bimanual finger postures are more difficult than
578 bimanual hand postures for some AD or FPN patients.

579 Two hypotheses predicted a body part effect. The body representation hypothesis
580 predicted a “Finger > Hand” dissociation, whereas the visuoconstructive hypothesis predicted
581 the reverse “Finger < Hand” dissociation. We observed more “Finger < Hand” dissociations,
582 which is more in line with the visuoconstructive hypothesis than with the body representation
583 hypothesis. The higher frequency of these dissociations in the FPN group (Table 5), as well as
584 the possible link to visuoconstructive impairments (Table 3), is also consistent with this
585 hypothesis. These findings are consistent with a causal effect of visuoconstructive abilities on
586 imitation performance. Previous studies have documented abnormal finger imitation in patients
587 with dementia with Lewy bodies, which cognitive profile is roughly characterized by
588 visuoconstructive and executive dysfunction (Kemp et al., 2017). Nagahama et al. (2015) have
589 found that AD patients scored higher than patients with dementia with Lewy bodies on
590 unimanual finger postures (96% and 83% of the maximum score, respectively), a pattern similar
591 to the AD/FPN differences we observed. They interpreted this finding as a consequence of
592 frontal-parietal cortical lesions associated with attentional, executive and visuospatial

593 impairments. Souza et al. (2016) have also found a correlation between finger imitation and
594 cognitive (visuospatial and executive) scores in patients with Parkinson’s disease. So, patients
595 from the FPN group probably had more difficulties with finger postures than with hand postures
596 because they had more important visuoconstructive deficits than other patients. This may be
597 explained by visual-spatial deficits following right parietal lesions and preventing patients from
598 extracting the visual model (especially in the light of positive correlations between the
599 visuoconstructive score and all imitation conditions). Another explanation could be that
600 visuoconstructive deficits following left parietal lesions prevented patients from selecting and
601 organizing the different subcomponents of finger postures. It should be noted that the
602 dissociations of interest, including “Finger < Hand” dissociations, were documented in each of
603 the three clinical groups, meaning that no dissociation can be considered pathognomonic. This
604 may reflect the complexity and multi-determined nature of imitation. Of note, many FPN cases
605 failed to imitate not only finger, but also hand postures, resulting in the absence of Finger/Hand
606 dissociation. Perhaps these patients had both visual-spatial and visuoconstructive deficits
607 (altering finger postures), and body representation deficits (altering hand postures) due to
608 widespread, bilateral lesions of the parietal lobes. Studies with larger numbers of participants,
609 and hence higher statistical power, will be able to decisively separate the relative contributions
610 of visuoconstructive and body representations in supporting imitation performance. Future
611 studies may also investigate the lesion patterns that are associated with different directions of
612 dissociations.

613 Our findings allow us to discuss other hypotheses sometimes invoked to explain visuo-
614 imitative apraxia. While the visuoconstructive hypothesis has endeavored to break down the
615 direct route for imitation (Goldenberg & Hagmann, 1997; Rothi et al., 1991), the recently
616 revived sensori-motor conversion hypothesis (Achilles et al., 2017; see also Geschwind, 1975)
617 assumes that hand postures are more sensitive to brain damage that also affects finger postures,

618 which boils down to a difficulty effect. Our findings of frequent “Finger < Hand” dissociations
619 are at odds with this hypothesis. They rather suggest that finger and hand postures call for
620 different cognitive processes, as previously proposed on the basis of research with individuals
621 who have had stroke (Goldenberg & Hagmann, 1997; Goldenberg & Strauss, 2002;
622 Goldenberg, 1999). It should be acknowledged however that Achilles et al. have focused on
623 functions of the left hemisphere only whereas we studied patients with typically bilateral
624 lesions, resulting in a likely influence of right hemisphere lesions. Finally, according to the
625 limited resource theory (Rumiati & Tessari, 2002; Tessari & Rumiati, 2002), finger imitation
626 may require more motor resources than proximal movements, and hence poor finger imitation
627 may arise from a deficit of motor programming. This hypothesis is based on the widespread
628 motor representation of finger movements in the parietal (Hoeren et al., 2014; Tanaka et al.,
629 2001), left frontal (Goldenberg & Karnath, 2006; Haaland et al., 2000; Tanaka & Inui, 2002),
630 and motor cortex (Rumiati & Tessari, 2002; Tessari & Rumiati, 2002). This could be an
631 important aspect of an account of why individuals with corticobasal syndrome, who typically
632 have frontal-parietal lesions and limb-kinetic apraxia, may show dissociations between finger
633 and hand configurations (Leiguarda et al., 2002; see also Holl et al., 2011). However, it does
634 not apply well to cases with posterior cortical atrophy, who do not have motor symptoms. Future
635 studies may help disentangle the motor and spatial components of imitation.

636 5.2. *THE BIMANUAL EFFECT*

637 Lesourd et al. (2013) have stressed a possible unimanual/bimanual dissociation in Alz-
638 heimer’s disease. Yamaguchi et al. (2010) have demonstrated that bimanual finger imitation
639 was more affected than unimanual finger imitation in this disease. Interestingly, Nagahama et
640 al. (2015) have found that unimanual finger imitation was more disrupted in patients with de-
641 mentia with Lewy bodies than in those with Alzheimer’s disease, while bimanual imitation was
642 affected to the same extent in both groups. Similarly, the FPN group had imitation impairments

643 with both unimanual and bimanual finger postures, whereas Alzheimer patients showed a se-
644 lective impairment of bimanual postures. This has two implications. First, impairment of uni-
645 manual imitation is likely to reflect atrophy of fronto-parietal areas, as already demonstrated in
646 stroke patients (Dressing et al., 2018; Goldenberg, 2009, 2017; Mengotti et al., 2013; see also
647 Tanaka et al., 2001; Tanaka & Inui, 2002). Second, bimanual imitation can be selectively im-
648 paired in (typical, amnesic) Alzheimer’s disease.

649 Nevertheless, some, but not all of the bimanual conditions were impaired in these patients.
650 We did not observe a higher prevalence of bimanual apraxia (Sanin & Benke, 2017). This may
651 be due to having experimentally separated multiple dimensions of the actions to be imitated
652 that modulate performance (asymmetry, midline crossing). Although bimanual apraxia has not
653 been clearly defined yet, it is logical to diagnose it in presence of normal unimanual imitation
654 but impaired bimanual imitation, otherwise patients should be diagnosed with apraxia but not
655 “bimanual” apraxia. Previous studies that have documented bimanual imitation impairments in
656 Alzheimer’s disease (Li et al., 2016; Sanin & Benke, 2017) did not use unimanual imitation as
657 a baseline, and studies that have directly compared uni- and bimanual imitation have used finger
658 postures only (Nagahama et al., 2015; Yamaguchi et al., 2010). This may have led to exaggerate
659 conclusions regarding bimanual apraxia – especially since we found “Finger < Hand” dissoci-
660 ations. Two of our findings do not confirm the prevalence of bimanual (visuo-imitative) apraxia
661 in AD: (1) the performance of most AD patients on bimanual hand subtests was actually normal
662 after controlling for body part, asymmetry, and midline-crossing (condition G in Figure 1); (2)
663 “Unimanual > Bimanual” dissociations were relatively rare in all groups, after controlling for
664 intrinsic task difficulty (using t-scores instead of raw scores).

665 In fact, “Unimanual < Bimanual” dissociations were unexpectedly frequent, a finding that
666 might reveal a facilitatory effect of the tactile feedback during bimanual imitation: Perhaps the
667 tactile feedback given by the contact of the left and right hands in the bimanual condition may

668 partially compensate for visuoconstruction/visuospatial deficits in the bimanual condition, in
669 some cases. Viewing unimanual actions as bimanual asymmetrical actions (since there is an
670 asymmetry of motor innervations during unimanual imitation) might also explain this dissoci-
671 ation. We compared the imitation of unimanual configurations to the imitation of bimanual,
672 symmetrical configurations (Table 5). Therefore, this dissociation between unimanual and bi-
673 manual imitation might, in fact, hide an effect of asymmetry. It is also possible that the need to
674 use one hand puts higher loads on right-left discrimination, which may be problematic for pa-
675 tients with posterior lesions. In any case, the need to use both hands to imitate the model was
676 not at stake in the poor performance of Alzheimer patients and hence, it cannot be concluded
677 that the latter have “bimanual” apraxia. In all likelihood, failure to imitate bimanual postures
678 arises from other dimensions of imitation tests, like asymmetry. The higher prevalence of bi-
679 manual imitation deficits in Sanin and Benke’s (2017) work (63% of maximum score, against
680 92% in the present study) might also indicate that movements, instead of static postures, add an
681 additional layer of difficulty to imitation tests for patients with Alzheimer’s disease (see also
682 Buxbaum et al., 2014 on posture versus kinematic components of imitation).

683 5.3. *THE ASYMMETRY EFFECT*

684 Asymmetrical postures were more difficult than their symmetrical equivalent for 11 cases
685 (i.e., 17% of the clinical sample), including 8 AD cases (i.e., 25% of AD cases). The same
686 dissociation has been documented in patients with Alzheimer’s disease or amnesic mild cog-
687 nitive impairment with finger postures (Li et al., 2016), as well as in healthy older adults (Bau-
688 mard et al., 2020). Why is asymmetry difficult for patients?

689 Two hypotheses predicted this pattern. The visuoconstructive hypothesis is unlikely given
690 that “Asymmetrical < Symmetrical” dissociations were found only in the SD and AD groups,
691 who had normal visuoconstructive skills. The findings are consistent with the goal-directed

692 theory of imitation (Bekkering et al., 2000, 2005; Gleissner et al., 2000) and may reflect a dis-
693 organization of hierarchical goal structure: The higher the number of subgoals (i.e., same con-
694 figuration for both hands in symmetrical postures, but different configurations in asymmetrical
695 ones), the higher the difficulty of the task for patients. That said, SD and AD patients did not
696 commit more symmetry errors than healthy controls, which weakens this hypothesis. An alter-
697 nate hypothesis could be that AD patients struggled with task complexity because of executive
698 dysfunction. Indeed, the number and ambiguity of visually similar elements logically increase
699 with asymmetrical postures, as compared to symmetrical ones. This presumably calls for high
700 executive control in the form of strategic analysis, planning and working memory, a necessary
701 condition for detecting minor left-right differences, sequencing and maintaining relevant sub-
702 goals temporarily (e.g., left hand, then right hand), and checking the quality of the production
703 (Luria, 1978; Luria & Tsvetkova, 1964). Based on the bimanual control system theory (Brink-
704 man & Kuypers, 1973; Kelso et al., 1979; Kinsbourne, 2003; Huhn et al., 2014), one could also
705 argue that patients had difficulties in desynchronizing hand movements, which boils down to a
706 deficit of inhibition of dominant patterns. Nevertheless, difficulties in inhibiting hand syn-
707 chrony should also have resulted in more symmetry errors in patients than in healthy controls,
708 which was not the case. This strengthens the hypothesis that planning and/or working memory
709 deficits, but not inhibition deficits, are at the root of visuo-imitative apraxia in Alzheimer's
710 disease, regarding finger postures at least. This "complexity account" of imitation may explain
711 why the performance of patients (but also of healthy controls) decreased as a function of the
712 number of effects combined into one and the same gesture (Table 4; e.g., bimanual+asymmetry
713 yield the most marked impairments). Of note, the Frontal Assessment Battery probably failed
714 to capture the abovementioned executive deficits properly as it does not assess working memory
715 and planning, a limitation that future studies would need to address.

716 5.4. *THE MIDLINE CROSSING INHIBITION EFFECT*

717 The “Crossed < Uncrossed” dissociation, as well as midline crossing errors, were ob-
718 served mainly in the group of patients with fronto-parietal brain lesions (FPN), in both cases
719 with posterior cortical atrophy, and cases with corticobasal syndrome. Using one crossed finger
720 configuration, Yamaguchi et al. (2010) have found significant imitation impairment in Alz-
721 heimer patients. By dissociating Alzheimer and FPN cases, we found that midline crossing was
722 actually more clearly an issue for the latter, with hand configurations at least. These findings
723 may support an association of midline crossing inhibition with cortical atrophy of fronto-pari-
724 etal areas, although a contribution from subcortical lesions is possible regarding cases with
725 corticobasal syndrome.

726 Two hypotheses predicted a midline-crossing effect. The goal-directed theory assumed
727 that midline crossing, a secondary subgoal, would be more sensitive to brain lesions than
728 higher-order goals (Bekkering et al., 2005; Gleissner et al., 2000). Goal-directed, contralateral
729 movements have been associated with increased fMRI signals in dorsal premotor areas (Koski
730 et al., 2002), a brain region frequently affected in CBS cases. The findings are therefore con-
731 sistent with this hypothesis, even more so as the FPN group committed more midline-crossing
732 errors than healthy controls. That said, if the performance was only a function of the number of
733 subgoals, then we should also have observed an “Asymmetrical < Symmetrical” dissociation in
734 the FPN group (see section 5.3). So, the goal-directed theory only partially accounts for the
735 results.

736 According to the body representation hypothesis, midline crossing inhibition may reflect
737 left-right disorganization (a possible role of asomatognosia, i.e. imperceptions of some parts of
738 the body, has been suggested; see Ajuriaguerra, 1960; Ajuriaguerra et al., 1966; see also Fein-
739 berg & Venneri, 2014). Since the ability to seamlessly cross the body midline reflects the in-
740 tegrity of body schema (Furlanetto et al., 2014; Goody & Reinhold, 1952; Yamamoto &
741 Kitazawa, 2001), midline-crossing inhibition may be interpreted as an impairment of the body

742 schema in the FPN group. The role of the left parietal lobe in left-right orientation has long
743 been demonstrated by cases of Gerstmann syndrome (for a review, see Rusconi et al., 2009;
744 Vallar, 2007), a syndrome that has also been described in PCA cases (Benson et al., 1988; see
745 also Tang-Wai et al., 2004). Interestingly, patients with pure forms of this syndrome do not
746 show problems in distinguishing left from right on their own body, but they do on the exam-
747 iner’s body. It turns out that establishing correspondence between left-right parameters of the
748 self and of the examiner is critical when it comes to imitation. To sum up, lesions of fronto-
749 parietal areas may disrupt the body schema, resulting in midline-crossing inhibition (see also
750 Wallon & Lurçat, 1962). It should be acknowledged that the body representation hypothesis
751 also predicted a “Hand < Finger” dissociation, while we found the reverse pattern in the FPN
752 group. This may suggest that body image (i.e., knowledge about the visual image of the body,
753 needed to imitate hand postures; Goldenberg, 1995) is more resistant to fronto-parietal brain
754 lesions than the body schema.

755 **6. LIMITATIONS OF THE STUDY**

756 This study was not without limitations. First, while we used classical coding systems to
757 make our study comparable to previous ones, the use of different coding systems for finger and
758 hand postures may have influenced the findings. Second, even though we included patients with
759 distinct diagnoses and cognitive profiles, patients with neurodegenerative diseases may have
760 multiple neuropsychological impairments (e.g., visual-spatial, visuoconstructive, body repre-
761 sentation deficits), resulting in the absence of dissociation between imitation conditions in some
762 cases. Third, future studies could explore imitation-cognition correlations using more specific
763 neuropsychological tests than in the present work, as well as larger samples. Future studies may
764 also incorporate measures of lesion extent. Fourth, we cannot rule out a possible effect of the

765 subtest and item sequence on the performance. For example, the imitation of asymmetrical con-
766 figurations always preceded the imitation of symmetrical ones, which might have contributed
767 to the overall better performance with the latter than with the former.

768 **7. CONCLUSION**

769 Different neurodegenerative processes affect fronto-parietal versus temporal lobe struc-
770 tures, resulting in different performance levels on tests of imitation. Patients with semantic de-
771 mentia had subnormal imitation skills, while patients with lesions to fronto-parietal areas (pos-
772 terior cortical atrophy, corticobasal syndrome) showed frequent and marked imitation deficits.
773 Only in this group was unimanual imitation impaired. By making a distinction between the
774 amnesic form of Alzheimer's disease, and posterior cortical atrophy, we found that visuo-imi-
775 tative apraxia was less marked in AD.

776 This study also demonstrated significant effects of imitation conditions, confirming the
777 need to further break down the direct route for imitation. "Finger < Hand" dissociations were
778 more frequent in patients with lesions of fronto-parietal areas, probably because of visuocon-
779 structive deficits. Furthermore, our findings did not confirm the prevalence of "bimanual
780 apraxia" in dementia, defined as a selective impairment of bimanual imitation. Difficulty rather
781 arises from the asymmetry of the model, which probably puts high demands on executive con-
782 trol and working memory. Finally, difficulties crossing the body midline were more frequent in
783 the FPN group, presumably reflecting body schema disorganization.

784 To conclude, our findings have implications for future clinically oriented research. First,
785 unimanual configurations may offer a basis to detect apraxic deficits in posterior cortical atro-
786 phy and corticobasal syndrome, but not Alzheimer's disease. Second, the finding of between-
787 task dissociations in 34/63 cases (54%) suggests that meaningless imitation is a complex task

788 tapping multiple processes. Performance in meaningless imitation is modulated by a number of
789 parameters of the imitated actions (midline crossing, asymmetry). The implication is that imi-
790 tation tests which include only a limited set of postures cannot properly assess patients' imita-
791 tion ability. The use of mixed lists of postures with variability in the body part, bimanual, asym-
792 metry, and midline-crossing dimensions, is recommended to avoid misestimating the severity
793 of apraxia in this clinical population. As the present study focused on meaningless imitation,
794 future works may directly compare meaningful and meaningless postures and gestures. Finally,
795 future studies may explore how the presentation of the postures (i.e., first person versus third
796 person) may impact the performance (Lesourd et al., 2017; Sugaya et al., 2022).

797

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1147 **DATA AVAILABILITY STATEMENT**

1148 The authors confirm that the data supporting the findings of this study are available within
1149 the article [and/or] its supplementary materials.

1150

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1151

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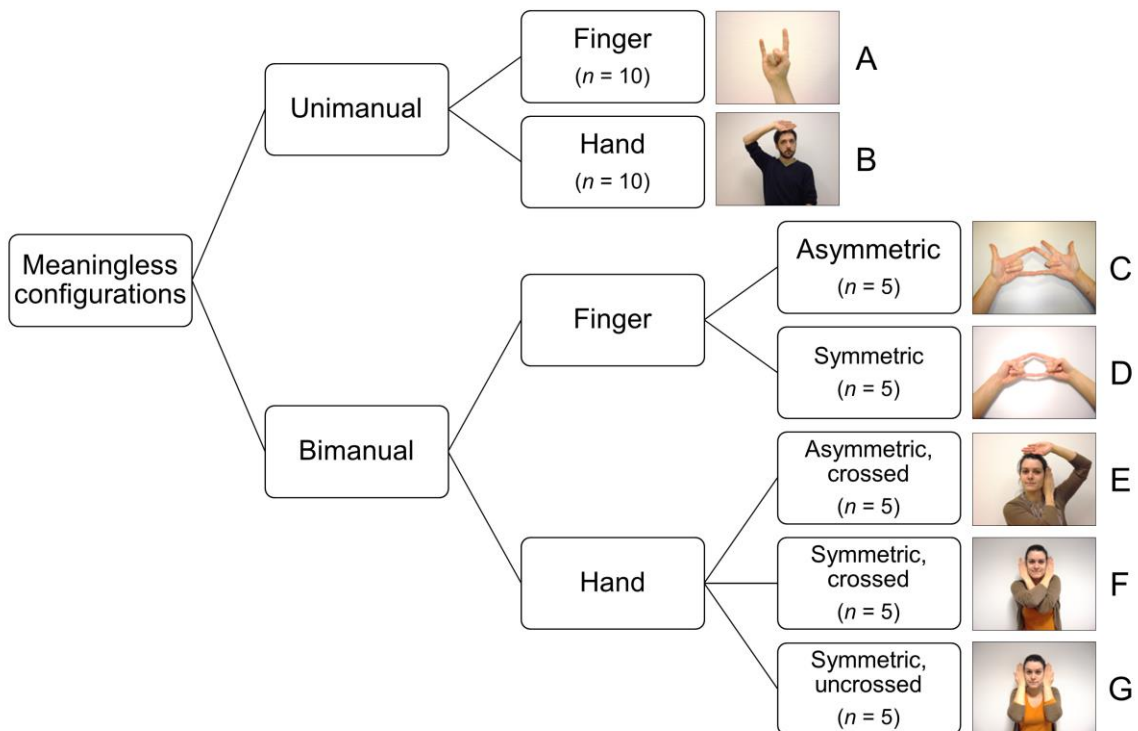
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1158

1159 **FIGURE 1. MEANINGLESS POSTURES**



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1161 *Notes.* The *n* values correspond to number of items. Pictures provide one example for each subtest. Different letters identify different subtests.

1162 Items were presented in the following order: A, C, D, B, E, F, G. Symmetrical and asymmetrical configurations were purposely equivalent so

1163 as to make them comparable. The same was true for crossed and uncrossed postures. The tasks were administered in this order: A, B, C, D, E,

1164 F, G.

1165

1166

TABLE 1. CORE HYPOTHESES AND PREDICTIONS

Hypothesis	Predicted effects
Body representation hypothesis. Imitation of hand or crossed configurations calls for tridimensional or left-right representation of the body (respectively).	(1) "Body part effect α" : Unimanual hand < unimanual finger. (2) "Midline crossing effect" : Bimanual hand symmetrical crossed < bimanual hand symmetrical uncrossed. Midline crossing errors should be observed.
Visuoconstructive hypothesis. Imitation, especially of finger postures, calls for visual-spatial skills and executive functions. The more complex the model, the more difficult the task.	(1) "Body part effect β" : Unimanual hand > unimanual finger. (2) "Bimanual effect α" : Bimanual finger (symmetrical) < unimanual finger + bimanual hand (symmetrical, uncrossed) < unimanual hand. (3) "Asymmetry effect" : bimanual asymmetrical finger < bimanual symmetrical finger + bimanual asymmetrical hand (crossed) < bimanual symmetrical hand (crossed). Positive correlation with visuoconstructive and/or executive scores.
Goal-directed theory. Imitation results from the combination of elementary action patterns (subgoals) into more general action patterns (goals). Impaired imitation of asymmetrical gestures (i.e., different goals for each hand) but not of symmetrical gestures (i.e., similar goals) may be interpreted as a disruption of the hierarchical goal structure. Midline crossing is represented as an additional subgoal and hence increases the difficulty of imitation.	(1) "Asymmetry effect" : same as above (except regarding correlations). (2) "Midline crossing effect" : same as above.

1167

Notes. Only the hypotheses that predicted opposite patterns of performance (referred to as " α " and " β ") are shown; see the text for details on

1168

other existing hypotheses. None of the hypotheses predicted "Symmetrical < Asymmetrical" or "Uncrossed < Crossed" dissociations.

1169

1170

1171 **TABLE 2. DEMOGRAPHIC AND CLINICAL DATA**

	HC group	SD group	AD group	FPN group
	N = 32	N = 16	N = 32	N = 15
Women/men	22/10	8/8	22/10	10/5
Age (M, (SD))	75.4 (6.4)	67.3 (7.4)	77.1 (7.4)	69.5 (6.1)
Years of education (M, (SD))	12.3 (4.7)	12.1 (2.8)	9.0 (4.3)	9.4 (5.1)
MMSE	27.3 (1.8)	22.8 (4.9)^a	20.1 (2.7)	18.8 (7.0)^c
FAB	-	12.8 (2.7) ^a	12.9 (2.4)	11.6 (3.7) ^b
BEC 96 Total	87.6 (5.3)	62.1 (14.1)^b	67.0 (8.8)	62.4 (20.6)^c
Working memory	11.7 (1.6)	10.8 (3.0)	11.5 (2.2)	10.0 (4.1)
Orientation	11.5 (1.0)	9.3 (3.5)	5.5 (3.9)	8.3 (4.2)
Verbal learning	10.7 (1.5)	6.8 (2.9)	6.6 (2.8)	6.9 (3.9)
Visual recognition	10.8 (1.0)	8.3 (2.7)	5.2 (2.3)	7.3 (2.9)
Verbal reasoning	9.1 (1.9)	6.7 (2.7)	8.7 (2.0)	6.3 (2.9)
Verbal fluency	11.7 (1.1)	4.9 (2.7)	9.6 (2.7)	9.3 (3.7)
Naming	11.4 (0.8)	4.6 (2.7)	10.2 (1.9)	9.1 (2.3)
Visuoconstructive skills	10.9 (1.4)	10.8 (2.2)	9.8 (2.7)	4.5 (4.9)

1172 *Notes. Data between brackets are standard deviations. Bold values are significant differences between patients and healthy controls. Missing*
 1173 *data due to comprehension impairments, depressive symptoms or scheduling issues: ^a n = 6; ^b n = 4; ^c n = 3. HC: Healthy controls; SD:*
 1174 *Semantic dementia; AD: Alzheimer's disease; FPN: Frontal-parietal network.*

1175

1176 **TABLE 3. SEVERITY OF IMITATION DEFICIT AND**
 1177 **RELATIONSHIP TO COGNITION**

N imitation conditions impaired	N cases			Cognitive scores		
	SD	AD	FPN	MMSE (0-30)	FAB (0-18)	BEC Visuoconstructive score (0-12)
0	11	17	2	21.0 (3.6)	13.6 (2.4)	10.4 (2.3)
1-2	5	7	3	22.1 (2.9)	12.3 (2.5)	9.6 (3.2)
3-4	0	8	0	18.9 (4.1)	12.1 (2.4)	9.0 (3.5)
5-6	0	0	7	19.0 (6.9)	11.5 (2.5)	6.0 (4.6)
7	0	0	3	15.0 (7.9)	10.3 (4.8)	0.0 (0.0)

1178 *Notes.* Values between brackets are standard deviations.

1179

1180

TABLE 4. GROUP PERFORMANCE ON IMITATION TASKS

Group	Unimanual		Bimanual					Total score
	Finger (A)	Hand (B)	Finger		Hand			
			Asymmetric (C)	Symmetric (D)	Asymmetric crossed (E)	Symmetric crossed (F)	Symmetric uncrossed (G)	
Mean score (Standard deviation)								
HC	98.4 (3.7)	97.3 (5.4)	58.4 (19.4)	82.2 (19.5)	80.6 (22.9)	92.8 (11.1)	93.1 (13.5)	88.8 (8.4)
SD	99.4 (2.5)	97.8 (4.1)	48.8 (28.3)	81.3 (23.1)	77.5 (23.2)	98.8 (3.4)	93.3 (6.2)	88.5 (7.4)
AD	96.4 (7.6)	96.4 (5.7)	24.8 (24.2)	64.4 (26.8)	58.0 (24.8)	86.8 (15.5)	92.0 (11.5)	79.1 (9.8)
FPN	60.7 (31.5)	74.6 (25.5)	16.0 (25.0)	28.0 (31.0)	30.0 (39.6)	53.3 (36.4)	67.3 (31.5)	50.6 (28.7)
N (%) patients with pathological score								
Cut-off	<91	<87	<19	<42	<34	<70	<66	<72
SD	1 (6)	0 (0)	3 (19)	2 (13)	1 (6)	0 (0)	0 (0)	0 (0)
AD	7 (22)	3 (9)	11 (34)	7 (22)	4 (13)	2 (6)	1 (3)	6 (19)
FPN	13 (87)	9 (64)	10 (67)	11 (73)	10 (67)	9 (60)	4 (27)	11 (73)
Mean t-scores (Standard deviation)								
SD	0.2 (0.7)	0.1 (0.8)	-0.5 (1.4)	0.0 (1.2)	-0.1 (1.0)	0.5 (0.3)	0.2 (0.5)	0.0 (0.9)
AD	-0.5 (1.8)	-0.2 (1.1)	-1.6 (1.2)	-0.9 (1.2)	-0.9 (1.1)	-0.5 (1.4)	-0.1 (0.8)	-1.1 (1.1)
FPN	-10.0 (8.4)	-4.1 (4.6)	-2.2 (1.3)	-2.7 (1.6)	-2.2 (1.7)	-3.5 (3.2)	-1.9 (2.3)	-4.5 (3.4)

1181 *Notes. Mean score: bold values are significant comparisons (p < .05) with the HC group (two-way ANOVA with Group x Condition, and post-*
 1182 *hoc Tukey HSD tests with p-value adjustment). N patients with pathological score: bold values are significant comparisons (p < .05) with the*
 1183 *SD group (Chi-square or Fisher tests with N NORMAL/N IMPAIRED and GROUP/GROUP factors). The SD group was used in this compar-*
 1184 *ison because SD patients had normal to subnormal performance, and because it allowed to address the ventral-dorsal hypothesis of imitation.*
 1185 *t-scores and cut-off scores were determined using Crawford and Garthwaite's method (Crawford & Garthwaite, 2002, 2005).*

1186

1187 **TABLE 5. BETWEEN-TASK DISSOCIATIONS AND LINKS TO COGNITION**

Effect	Dissociation	N cases			Cognitive scores		
		SD (n = 16)	AD (n = 32)	FPN (n = 15)	MMSE (0-30)	FAB (0-18)	BEC – Visuoconstru ctive score (0- 12)
Body part	Finger < Hand ^a	1	5	7	17.5 (5.3)	11.1 (2.2)	4.9 (4.6)
	No dissociation	15	27	8	21.1 (4.0)	13.1 (2.7)	10.0 (2.9)
Bimanual	Unimanual < Bimanual ^b	1	3	10	16.6 (5.3)	10.6 (3.2)	3.8 (3.7)
	Unimanual > Bimanual ^c	2	5	0	21.0 (2.3)	12.5 (2.6)	9.3 (3.7)
	No dissociation	13	24	5	21.2 (4.1)	13.3 (2.4)	10.3 (2.7)
Symmetry	Asymmetrical < Symmetrical ^d	3	8	0	21.4 (4.2)	13.0 (2.3)	10.8 (1.6)
	Asymmetrical > Symmetrical ^e	0	1	2	19.0 (4.2)	10.0 (2.5)	3.7 (1.5)
	No dissociation	13	23	13	20.1 (4.6)	12.8 (3.4)	8.7 (3.6)
Midline crossing	Crossed < Uncrossed ^f	0	4	7	19.4 (5.6)	11.8 (3.6)	7.4 (5.0)
	No dissociation	16	28	8	20.5 (4.3)	12.8 (2.5)	9.1 (3.7)

1188 *Notes.*

1189 a Dissociations: unimanual finger < unimanual hand, or bimanual finger symmetrical < bimanual hand symmetrical uncrossed postures. For n = 1 case, the pattern was inconsistent between uni- and bimanual
1190 postures so it was decided to include him in the “Finger < Hand” subgroup to avoid over representing an atypical pattern.

1191 b Dissociations: unimanual finger < bimanual finger symmetrical, or unimanual hand < bimanual hand symmetrical uncrossed postures.

1192 c Dissociation: unimanual finger > bimanual finger symmetrical postures.

1193 d Dissociations: bimanual finger asymmetrical < bimanual finger symmetrical, or bimanual hand asymmetrical crossed < bimanual hand symmetrical crossed postures.

1194 e Dissociation: bimanual hand asymmetrical crossed > bimanual hand symmetrical uncrossed.

1195 f Dissociation: bimanual hand symmetrical crossed < bimanual hand symmetrical uncrossed.

1197 **TABLE 6. MULTIPLE REGRESSION ANALYSIS**

Predicted variable	Significant predictive variables	F (df)	Adjusted R ² (explained variance)	P-value
Unimanual Finger	Visuoconstruction	18.4 (3,43)	0.53	< .001
Unimanual Hand	MMSE + visuoconstruction	14.0 (3, 42)	0.47	< .001
Bimanual Finger Asym.	Visuoconstruction	4.3 (3, 43)	0.17	.010
Bimanual Finger Sym.	Visuoconstruction	8.8 (3, 43)	0.34	< .001
Bimanual Hand Asym. Crossed	Visuoconstruction	6.2 (3, 43)	0.25	.001
Bimanual Hand Sym. Crossed	Visuoconstruction	9.8 (3, 43)	0.36	< .001
Bimanual Hand Asym. Uncrossed	MMSE + visuoconstruction	11.2 (3, 43)	0.40	< .001

1198 *Notes.* Multiple linear regression performed on the whole clinical sample (without healthy controls), testing how the MMSE, visuoconstructive, and executive (FAB)
 1199 scores predicted the imitation scores. “df”: degrees of freedom.

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